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Anatomic Correction of Transposition of the Great Arteries in Neonates

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Objectives. **This retrospective study attempts to assess the results of the neonatal anatomic repair of transposition of the great arteries by a single institution.**

Background. **Anatomic correction of transposition of the great arteries by means of the arterial switch operation is now widely accepted as the therapeutic method of choice.**

Methods. **Four hundred thirty-two consecutive neonates underwent an arterial switch operation for various forms of transposition of the great arteries. There were 362 neonates with transposition and intact ventricular septum, 47 with a ventricular septal defect, 6 with intact ventricular septum and coarctation of the aorta and 17 with ventricular septal defect and coarctation. Among patients with coarctation, 18 underwent a single-stage repair through median sternotomy. The mean age was 13.1 ± 4.2 days. Coronary artery distribution was described according to the origin and initial course of the arteries.**

Results. **Overall in-hospital mortality was 7.8% (34 patients) and was 7.6% for transposition with intact ventricular septum, 8.5% for transposition with ventricular septal defect and 13.3% for transposition with ventricular septal defect and coarctation. Univariate analysis of risk factors revealed that coronary anatomy was the main determinant for operative survival. A mean follow-up time of 44 ± 19 months was achieved in all but five survivors. More than 95% were in New York Heart Association functional class I, without medication and with normal left ventricular function. Reoperation was performed in 20 patients: early reoperation (<30 days) in 4 and late reoperation in 16. Actuarial survival rates at 5 years were 91.3% (transposition with intact ventricular septum) and 81.06% (transposition with ventricular septal defect). As well, freedom from reoperation at 5 years was 96.8% (transposition with intact ventricular septum) and 84.6% (transposition with ventricular septal defect).**

Conclusions. **The arterial switch operation is feasible in almost all forms of transposition of the great arteries in neonates as primary and definitive repair. Palliative surgery is recommended in cases with complex intracardiac anatomy not amenable to early repair.**

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Complete transposition of the great arteries is one of the most common cardiovascular anomalies. Proposed procedures for surgical repair have focused on the problem at different anatomic sites: the great veins (1), the atria (2,3) or the great arteries (4). Arterial repair theoretically seems to be a better option because it does not introduce any additional intracardiac anomalies, and it restores the left ventricle to its natural systemic function. However, anatomic correction is a delicate operation, necessitating transfer of the coronary arteries and reconstruction of the pulmonary artery. Several pioneer attempts were experimentally performed in the early days of open heart surgery (4–7). All of these ingenious procedures failed because the coronary arteries were not involved, or were only partially involved, in the switching maneuver.

In 1961, Idriss et al. (8) reported attempting anatomic correction in two children in whom a circumferential rim of the aorta, together with the cororary ostia, was resected and rotated onto a new site over the transected pulmonary artery, followed by switching the great vessels. In their attempts, there was no survival because of left ventricular failure.

Progress in cardiopulmonary bypass technology, microsurgery with optical assistance and knowledge of physiopathology led to the first successful arterial switch procedure by Jatene et al. (9) in 1975. The presence of a ventricular septal defect in this case contributed to maintenance of suitable left ventricular function for supporting the systemic work load immediately after repair. In 1984, Castaneda et al. (10) successfully extended this repair to newborns with transposition of the great arteries, believing that the left ventricle of these patients should be well suited for the procedure, because it supported systemic pressure during gestation.

Encouraged by the successful report of the Boston group, in April 1984 we decided to attempt to treat all forms of transposition by anatomic correction in the neonatal period.

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Table 1. Clinical Characteristics of 432 Neonates With Transposition of the Great Arteries

	n	Age (days)	Weight (kg)
TGA-IVS	362	8.5 ± 4.0	3.3 ± 0.5
TGA-VSD	47	18.5 ± 12	3.0 ± 0.2
TGA-IVS-CoA	6	8.1 ± 4.5	3.0 ± 0.1
TGA-VSD-CoA	17	10.2 ± 3.4	3.2 ± 0.1
Total	432	13.1 ± 4.2	3.2 ± 0.3

Data are expressed as number of patients or mean value ± SD. CoA = coarctation of the aorta; IVS = intact ventricular septum; TGA = transposition of the great arteries; VSD = ventricular septal defect.

We report herein our total experience with such anatomic correction in neonates.

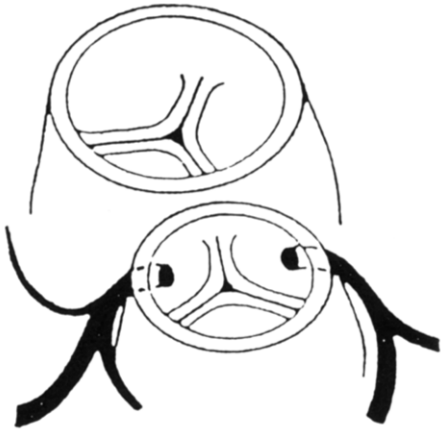

Figure 1. Type I coronary artery patterns. Normal course of right and left coronary arteries.

Methods

Patients (Table 1). From April 1984 to April 1992, 432 consecutive neonates underwent anatomic repair of various forms of transposition of the great arteries at Marie Lannelongue hospital. Three hundred sixty-two patients had simple transposition, 47 had transposition and a large ventricular septal defect, 6 had transposition with intact ventricular septum and coarctation of the aorta and 17 had transposition with ventricular septal defect and coarctation. In patients with transposition and intact ventricular septum, the mean age was 8.5 ± 4 days, for those with transposition and ventricular septal defect, it was 18.5 ± 12 days. The mean weight for the entire series was 3.3 ± 0.5 kg. Ninety-two percent of all patients were operated on within the 1st 2 weeks of life (Table 1).

Preoperative management. A balloon atrial septostomy was performed in 375 patients (86.8%), 339 with simple transposition of the great arteries and 36 with transposition of the great arteries and ventricular septal defect. Prostaglandin E_1 infusion (0.025 μg/kg body weight/min) was necessary to maintain a stable hemodynamic balance in 371 patients (85.8%), 348 with simple transposition of the great arteries and 23 with transposition of the great arteries and ventricular septal defect.

At the start of the study, all patients underwent cardiac catheterization; however, with increasing experience in Doppler echocardiographic studies, most of the anomalies were well diagnosed by this technique, and at present, only 10% of patients undergo left heart catheterization; the others have an atrial septostomy only when necessary, with no further angiographic study.

Preoperative left ventricular function was evaluated by two-dimensional echocardiography. Left ventricular geometry was assessed by a subxiphoid transverse view by measuring in end-systole the ratio of the lateral/anteroposterior diameter of the left ventricle. Therefore, the septal curvature gave sufficient information concerning the ability of the left ventricle to support the systemic work load immediately after the operation. This was considered a rough but reliable estimation of left ventricular systolic pressure (11).

Anatomic findings. In the group with simple transposition of the great arteries, six patients presented with associated

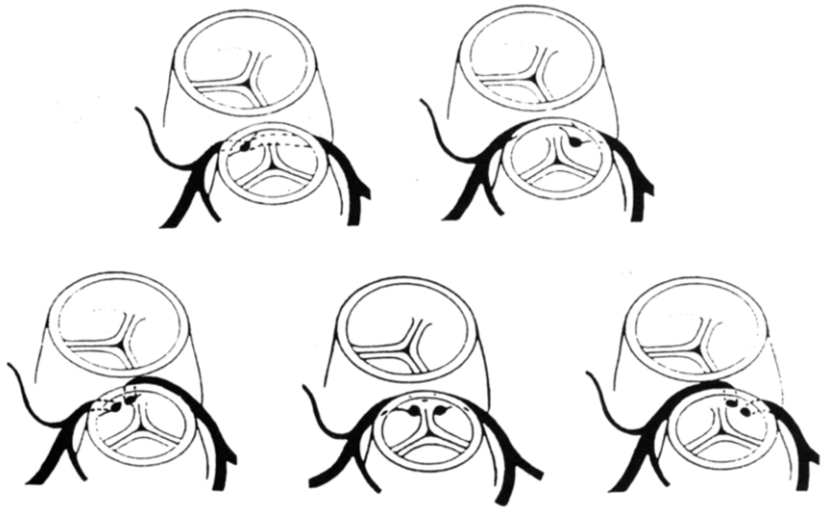

Figure 2. Type II coronary artery patterns. One or both arteries course between the great vessels (five subtypes).

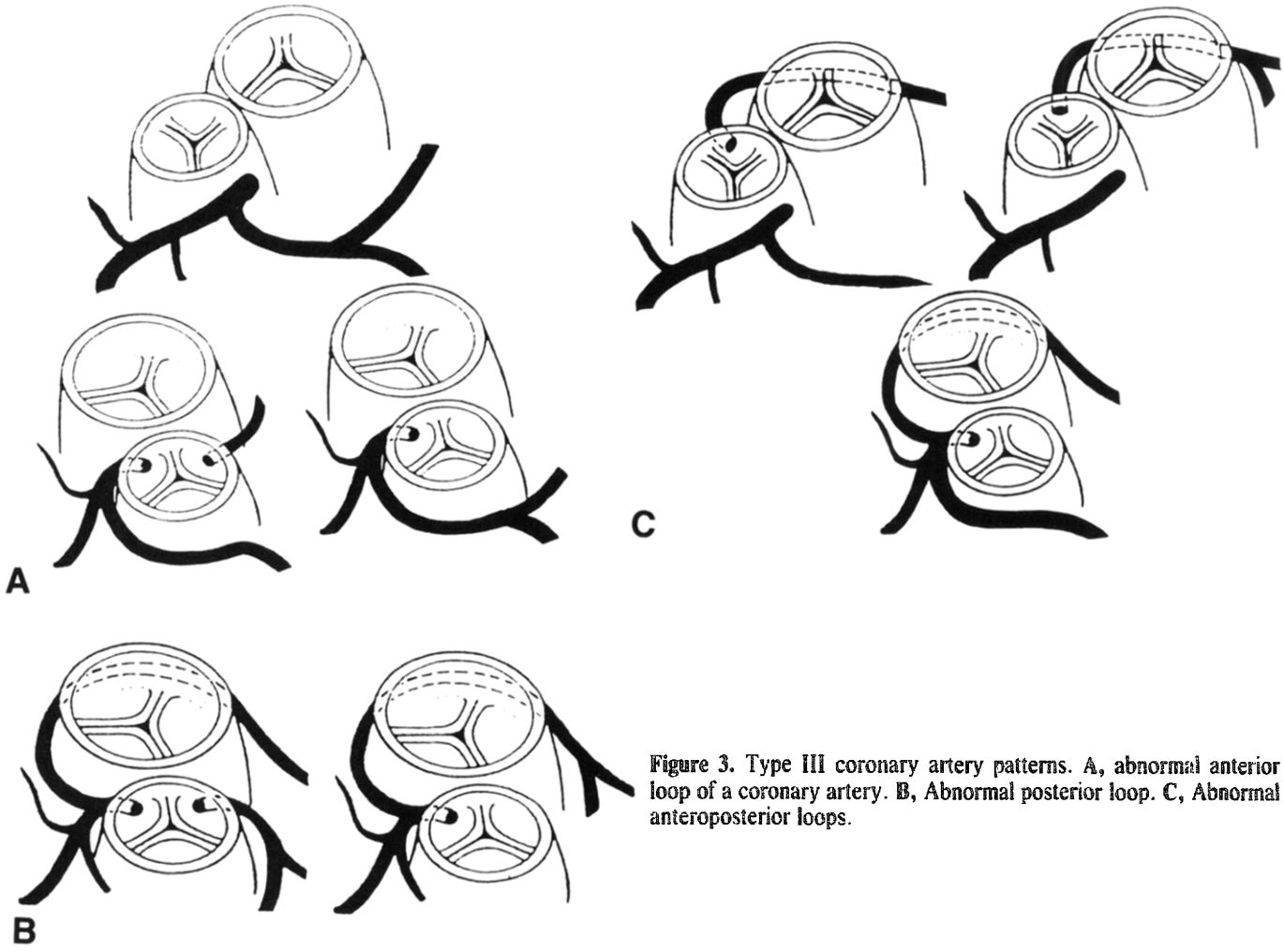

Figure 3. Type III coronary artery patterns. A, abnormal anterior loop of a coronary artery. B, Abnormal posterior loop. C, Abnormal anteroposterior loops.

coarctation of the aortic isthmus. In the group with transposition and ventricular septal defect, 17 patients had associated aortic coarctation with a hypoplastic transverse arch, and 3 patients had multiple ventricular septal defects. In this group, subaortic stenosis was present in four patients.

Dextrotransposition was present in 402 patients (93%): 344 with simple transposition and 58 with transposition and ventricular septal defect. In 30 patients (6.9%), the great arteries were side by side, the aorta being located on the right. The diameters of the aorta and pulmonary artery were similar in patients with simple transposition, whereas the pulmonary trunk diameter was 1.5 times larger than that of the aorta in 36 patients with transposition and ventricular septal defect. This discrepancy was more pronounced in patients with associated aortic coarctation.

Coronary artery patterns were classified differently from the usual procedure. This classification takes into account the origin and initial course of the coronary arteries, which are the main determinants for the mechanisms of myocardial ischemia after relocation. Table 2 shows the four coronary patterns observed. At no time was the arterial switch procedure abandoned for a Senning procedure because of complex coronary anatomy.

The location of the ventricular septal defect was assessed preoperatively by echocardiography and by intraoperative analysis. It was perimembranous in 42 patients and was located in the trabecular septum in 13 patients and in the infundibular septum in 5. Four patients presented with a subpulmonary ventricular septal defect and conal malalignment (Taussig-Bing malformation). In addition to these four, nine of the patients with transposition and ventricular septal defect had obvious malalignment of the outlet septum.

The anatomy of the aortic arch was normal in 409 patients. Twenty-three (5.3%) had a hypoplastic aortic arch (n = 16), an isthmic coarctation (n = 5) or an interrupted aortic arch (n = 2).

Surgical Technique

General procedures for cardiopulmonary bypass and surgical technique have been described elsewhere (12). In patients with transposition of the great arteries and ventricular septal defect, the septal defect was closed first. The right atrial approach was always attempted, but it was sufficient in only 36 patients. In the others, a right ventriculotomy (n = 13) or a transpulmonary approach (n = 15) was

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Table 2. Classification of Coronary Arteries in 432 Neonates With Transposition of the Great Arteries

Type	Description	Patients (no. [%]) All	TGA-IVS	TGA-VSD
I	Two ostia from posterior-facing sinuses and normal course of right and left coronary arteries (Fig. 1)	309 [71]	264	45
II	One or two ostia from one or two posterior-facing sinuses (five subtypes) but one or both arteries coursing between the great vessels (Fig. 2)	30 [6.9]	28	2
III	One or two ostia arising from one or two posterior-facing sinuses (seven subtypes), but either one of the coronary artery loops in front of the aorta, or the left coronary artery or left circumflex artery, or two coronary arteries loop around the great vessels (Fig. 3)	92 [21.3]	74	18
IV	Various combinations of types I to III	1 [.002]	0	1

Abbreviations as in Table 1.

necessary to secure closure of the septal defect. The arterial switch procedure was carried out as for simple transposition.

The proximal neopulmonary trunk was always reconstructed with a fresh pantaloon patch of autologous native pericardium. The pulmonary anastomosis was completed during rewarming.

Mean (±SD) aortic cross-clamp time was 67 ± 12 and 83.3 ± 20.5 min in patients with simple transposition and transposition with ventricular septal defect, respectively. Mean cardiopulmonary bypass time was 157 ± 33 and 170.6 ± 35.6 min in the respective groups.

In 18 patients with aortic coarctation (15 with and 3 without a ventricular septal defect), repair was performed in a single-stage procedure through a median sternotomy. The procedure began with aortic reconstruction under circulatory arrest and, after return to hypothermic perfusion, ended with closure of the septal defect and the arterial switch procedure.

Postoperative management was based on the same previously described principles. Routine use of bidimensional echocardiography in the intensive care unit allowed occasional changes in postoperative management.

All but five survivors were followed up. Follow-up data were obtained by letters and telephone calls to families and the patient's pediatric cardiologist.

Postoperative evaluation. Electrocardiograms (ECG), chest X-ray films, two-dimensional echocardiography and Doppler studies were performed every 6 months during the first postoperative year and annually thereafter. When these studies revealed any abnormalities, either 24-h Holter ambulatory ECG recording for rhythm disturbances or cardiac catheterization on the right and left sides was performed. Mean follow-up time was 44 ± 19 months.

Statistical analysis. Comparisons were appropriately performed by chi-square, Fisher or Student *t* tests. Time-related events were examined by the Kaplan-Meier actuarial method. Ratios are expressed with 95% confidence limits.

Results

Mortality. *Early deaths (Table 3).* There were 34 early deaths (7.8%) (95% confidence limits [CL], 0, 10.9%). In the group with simple transposition, mortality was 7.6% (95% CL 0, 11.1%, n = 28), and in the group with transposition and ventricular septal defect, it was 8.5% (95% CL 0, 21.2%; n = 4). Among patients who underwent one-stage repair of transposition with ventricular septal defect and coarctation of the aorta, there were two deaths (13.3%) (95% CL 0, 41.6%). The causes of death are listed in Table 3.

Univariate analysis of several factors, including age, weight, gender, preoperative status (septal curvature, acidosis, aortic saturation), coronary patterns, presence of ventricular septal defect, presence of coarctation, reveals that mortality was significantly higher in type II coronary patterns ($p < 0.01$).

Late deaths (Table 4). There were eight late deaths. In the group with simple transposition, two patients with unremarkable preoperative and postoperative courses died within 6 months of operation because of myocardial ischemia. Two other patients with simple transposition developed a superior vena cava thrombosis early after operation; however, with conservative management they were discharged from the hospital in normal condition. Unfortunately, both died at home 3 months later. One patient with

Table 3. Causes of Operative Mortality

	TGA-IVS	TGA-VSD	TGA-VSD-CoA
Coronary artery–related	16	2	1
Myocardial protection	4	—	—
LV dysfunction	4	—	—
Hemorrhage	4	2	1
Total	28 (7.6%)	4 (8.5%)	2 (13.3%)

Data are expressed as number (%) of patients. LV = left ventricular; other abbreviations as in Table 1.

Table 4. Causes of Late Death

	TGA-IVS	TGA-VSD
Myocardial infarction	2	1*
SVC thrombosis	2	1
Obstructive pulmonary vascular disease		1
Multiple VSDs		1

*Death occurred after heart transplantation. Data indicate number of patients. SVC = superior vena cava; other abbreviations as in Table 1.

transposition and ventricular septal defect died 6 months later of obstructive pulmonary vascular disease. Finally, three patients with transposition and ventricular septal defect and coarctation died of secondary causes. One had a nonfatal myocardial infarction after the arterial switch procedure. He underwent heart transplantation 3 years later in another institution and died soon after of acute graft rejection. Another developed a superior vena cava syndrome due to extrinsic compression. Reoperation was performed 3 years later and he underwent angioplasty of the superior vena cava. This patient died 2 years later from arrhythmias. In the last patient, multiple ventricular septal defects were overlooked. He underwent reoperation 3 months later and died of acute right ventricular failure.

Functional status of survivors. All but five survivors were followed up by their referring pediatric cardiologists. The length of follow-up ranged from 1 to 110 months (mean 44 ± 19). All were in New York Heart Association class I without medication.

Electrocardiogram. Ninety-seven percent of survivors had normal sinus rhythm. In only one patient did the ECG show ST–T changes in lateral leads. All patients with associated ventricular septal defect had right bundle branch block.

Twenty-four–hour Holter recordings were performed in 26 patients and showed minimal paroxysmal supraventricular arrhythmias in 4. Two patients had atrioventricular block that required a permanent pacemaker.

Left ventricular function. At the final echocardiographic examination, left ventricular dimensions and shortening fraction were within normal range in 95% of survivors. Five percent of patients had mild hypokinesia (shortening fraction <30%) or left ventricle enlargement, or both.

Ventricular outflow tracts. Right and left ventricular outflow tracts were assessed by color Doppler studies. The mean gradient between the right ventricle and the pulmonary artery was 11 ± 9 mm Hg. It was >30 mm Hg in 28 survivors with simple transposition and in 4 with transposition and ventricular septal defect.

No gradient was found on the left ventricular outflow tract. Aortic regurgitation was demonstrated in 30 patients (7.6%) (95% CL 0, −10.9%); 24 with simple transposition and 6 with transposition and ventricular septal defect. Microvalvular regurgitation was not considered in this series and was grade 2/6 in 23 patients, grade 3/6 in 5 and grade 4 in 2. One of the latter patients underwent reoperation and had an aortic valvuloplasty; however, all of the others remain asymptomatic and do not need medication.

Table 5. Early and Late Reoperations

Reason for Reoperation	TGA-IVS	TGA-VSD
Early Reoperation (<30 days)		
SVC thrombosis	2	
Aortic CoA	1	
Pulmonary stenosis		1
Late Reoperation (<30 days)		
RV outflow tract obstruction	3	2
Neo CoA	3	0
Recurrent CoA	1	0
Residual VSD	0	1 + 1*
Aortic regurgitation	1	0
Heart transplantation	0	1*
SVC syndrome	0	1*

*Late death. Data indicate number of operations. RV = right ventricular; other abbreviations as in Tables 1 and 4.

Reoperation (Table 5). Twenty patients (5.0%) (95% CL 0, −7.8%) required reoperation, and there were two early and one late death. *Early reoperation* (<30 days) was performed in four patients. Two had superior vena cava thrombosis and underwent emergency thrombectomy. One had an undiagnosed aortic coarctation; the other with transposition and ventricular septal defect presented postoperatively with a severe obstruction on the pulmonary artery reconstruction. *Late reoperation* was performed in 16 patients. Three with simple transposition underwent reoperation for right ventricular outflow tract obstruction. The mean right ventricular–pulmonary artery gradient before reoperation was 70.7 ± 12 mm Hg. A fourth patient with simple transposition underwent reoperation 6 years after the arterial switch procedure for aortic regurgitation. One other patient with simple transposition and aortic coarctation underwent reoperation for recurrent coarctation. In three patients with simple transposition, neoaortic coarctation appeared 3 to 6 months after the arterial switch operation. Two patients with transposition and ventricular septal defect underwent reoperation for right ventricular outflow tract obstruction and one for a residual septal defect. Patients with transposition and ventricular septal defect and coarctation underwent five reoperations: heart transplantation, superior cava enlargement and residual multiple ventricular septal defects, with two early deaths and one secondary death. Two other patients underwent reoperation for recurrent coarctation.

Actuarial survival rates at 5 years were separately analyzed according to the presence of a ventricular septal defect (Fig. 4). In patients with transposition of the great arteries with intact ventricular septum, the rate was 91.3 ± 0.3%; in those with transposition and a ventricular septal defect, it was 81.06 ± 8.5%. This difference was statistically significant (p = 0.01) by log-rank test. Freedom from reoperation at 5 years was also assessed according to the presence of a

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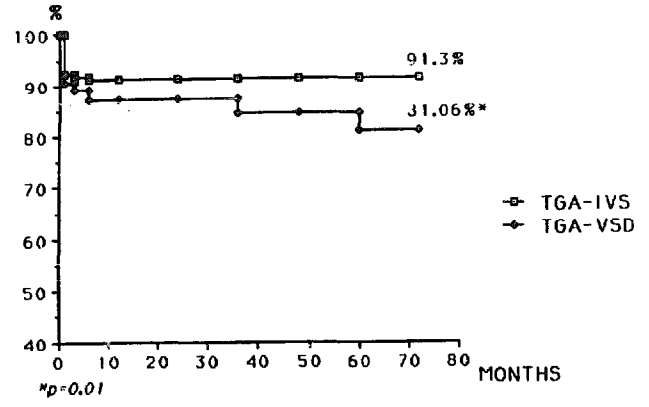


Figure 4. Actuarial survival. TGA-IVS = transposition of the great arteries with intact ventricular septum, including patients with coexisting coarctation of the aorta; TGA-VSD = transposition of the great arteries with ventricular septal defect, including patients with coexisting coarctation of the aorta.

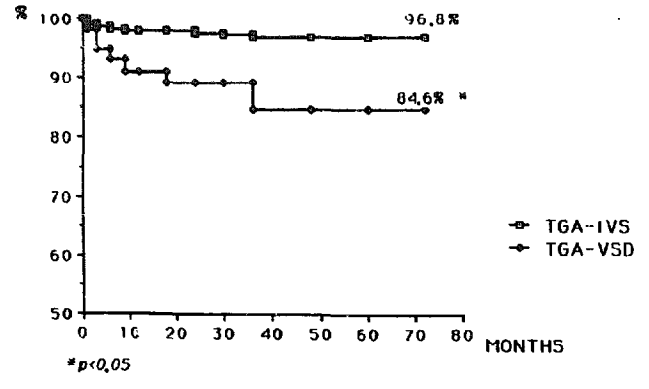
ventricular septal defect (Fig. 5). In simple transposition, it was 96.8 ± 2.4%; in transposition with ventricular septal defect, it was 84.6 ± 11.2%. This difference was also statistically significant ($p < 0.05$).

Discussion

This study describes one of the largest series of anatomic correction for various types of transposition of the great arteries during the neonatal period from a single institution. The overall survival rate for patients with transposition entering an intraatrial repair protocol should be estimated at approximately 70% to 80% at 5 years when considering the preoperative and postoperative deaths (13). These survival rates are much lower in patients with complex transposition of the great arteries with ventricular septal defect (14), with or without aortic coarctation.

Success in anatomic repair depends on two principal variables: preoperative left ventricular function and the quality of the operation. Immediately after operation, the left ventricle has to eject a normal cardiac output at systemic pressure in the aorta. This is the case in the neonatal period because during fetal life, pulmonary artery and aortic pressures are equal. In simple transposition, after birth, with the decrease in pulmonary vascular resistance and constriction of the ductus arteriosus, pulmonary artery and left ventricular pressures decrease dramatically to less than one third of systemic pressure. As a result, the left ventricle is not stimulated for growth adaptation and becomes (like the right ventricle in normal heart) thin and less contractile and more compliant. There is little doubt, from several centers including ours (15–17), that during the 1st 2 to 4 postnatal weeks, the left ventricle is still capable of sustaining a systemic work load. After this delay, the ability of the left ventricle is in greater jeopardy and depends greatly on the loading conditions. At the present time, we recommend performance of the arterial switch procedure within the 1st 2 weeks of life. In patients treated between the 2nd and 4th postnatal weeks, we have never experienced any operative failure due to inability of the left ventricle to support the systemic work load; however, most of the these patients needed longer postoperative inotropic support.

Figure 5. Freedom from reoperation. Abbreviations as in Figure 4.



After the neonatal period, several investigators (18,19) have recommended a two-stage approach, including as a first stage banding of the pulmonary artery, associated with a modified Blalock shunt "to prepare" the left ventricle, followed more or less rapidly by the arterial switch procedure. These investigators (18–21) described several indexes to optimize the timing for the arterial switch; however, although not included in the present series, four patients aged 2 months, each with transposition and intact ventricular septum underwent the arterial switch operation at our institution without left ventricular preparation and without death. This experience led us to question the exact indication for left ventricular preparation and the underlying mechanisms. In cases of transposition of the great arteries and ventricular septal defect, the suitability of the left ventricle is not questioned; however, we recommend anatomic repair within the 1st 4 postnatal weeks and before further development of pulmonary vascular hypertensive disease. Moreover, we have found that the sooner the arterial switch is performed in this subset, the smaller the size mismatch between the great arteries.

Finally, in patients with transposition of the great arteries and ventricular septal defect and coarctation of the aorta, anatomic repair should be performed as soon as possible, provided that the intracardiac anatomy allows complete repair.

Surgical difficulties. *Relocation of coronary arteries.* Many anatomic studies (22–26) have dealt with the anatomy of the coronary arteries; however, these attempts at classification were either incomplete or too complex for decision-making with regard to coronary transfer. On the basis of our experience, we propose a revised classification of coronary artery patterns that takes into account the origin and initial course of the artery. Three main types of coronary patterns are described (see Methods), which are closely related to the mechanism of ischemia after coronary transfer. Type I is the most common form. Coronary relocation results in backward translocation of the two coronary arteries, which can

be accomplished with minimal risk of deformation. Type II is characterized by coronary arteries originating and coursing between the aorta and the pulmonary artery. Coronary relocation requires turning the ostial patch upside down, which creates a risk of torsion. Type III corresponds to coronary arteries with an abnormal origin of one artery that runs transversely either in front of or behind the great vessels. In these cases, relocation may lead to stretching or kinking of the coronary vessels. Knowledge of the mechanisms of ischemia after relocation has led to a reduction in postoperative mortality from 40% in the first 10 cases to 7.8%.

Closure of the ventricular septal defect. Closure of the ventricular septal defect remains a challenging problem in neonates because tissues are very friable, and accurate closure requires perfect exposure of the defect. Moreover, in >20% of patients there is a malalignment of the conal components. In these patients, repair involves more rerouting of the left ventricular bloodstream to the pulmonary artery than a simple closure of the defect. Either a transventricular or a transpulmonary approach is preferred in these cases.

Concomitant repair of aortic arch anomalies. Our previous experience with two-stage repair in patients with transposition and ventricular septal defect and coarctation of the aorta was not satisfactory because of cumulative morbidity and mortality. This led us to consider one-stage repair. The aortic arch repair was performed as for interrupted aortic arch.

The appearance of a secondary neoaortic coarctation was a striking feature after anatomic repair of simple transposition of the great arteries. This was previously reported by Muster et al. (27), who related it to the Lecompte maneuver; however, all of the patients in the present series underwent this maneuver, and the incidence of aortic coarctation remained very low. One other explanation could be that during ductus division, some ductal tissues are invaginated into the aortic lumen, thus causing secondary coarctation.

Superior vena cava thrombosis occurred in five patients. Symptoms appeared within the 1st 2 postoperative days. In the beginning, conservative management based on heparinization was adopted; however, all of the patients who entered into this protocol finally died. A more aggressive approach was therefore adopted, and surgical thrombectomy was performed in two patients. Both of these patients survived and did not present any sign of superior vena cava syndrome at follow-up. We therefore recommend a very aggressive surgical approach for this complication as soon as symptoms occur.

Morbidity differs among the different types of transposition complexes. In simple transposition of the great arteries, late deaths occurred within the 1st postoperative year; thereafter, the survival curve reached a plateau. The reoperation rate was also low, and the main cause of reoperation was the occurrence of pulmonary stenosis; however, prompt follow-up of aortic valve function seems to be important because of the increasing occurrence of aortic valve incompetence.

Although operative mortality did not differ significantly in patients with transposition and ventricular septal defect, the group had four late deaths and significantly lower survival rates than those of patients with intact ventricular septum. This difference can be explained by two main factors: the spontaneous development of obstructive pulmonary vascular disease, as in one patient, and the complexity of repair, particularly when coarctation and banding of the pulmonary artery have previously been done. Two secondary deaths occurred in such patients. Conversely, the reoperation rate was also higher.

As we reported previously (28), right ventricular outflow tract obstruction is the main postoperative problem, particularly in patients with aortic coarctation and a small neopulmonary anulus and in patients with previous pulmonary artery banding. However, compared with patients with transposition and ventricular septal defect who were >1 month old, this group demonstrated no or minimal risk of obstructive pulmonary vascular disease, almost no size discrepancy between the great arteries and no need of a palliative operation. Among patients with transposition and ventricular septal defect and aortic coarctation who underwent single-stage repair, mortality and morbidity were higher; however, with increasing experience this rate tended to be lower.

Some follow-up studies have revealed the occurrence of neoaortic valve dysfunction in 5% to 50% of cases after the arterial switch operation (29). The present study confirms this observation with a somewhat lower rate that can be explained by the exclusion of microaortic regurgitation; however, one of the first patients who underwent an arterial switch operation underwent reoperation for severe aortic valve regurgitation. At operation, there was a huge dilation of the noncoronary Valsalva sinus, with a prolapse of the corresponding leaflet.

Finally, ventricular function in the survivors remains within the normal range and is comparable to that in other reports (15). All of the children can maintain a normal childhood without medication and with normal psychomotor development.

Conclusions. The arterial switch operation seems to be feasible in all forms of transposition of the great arteries as a primary and definitive procedure in neonates. Improvements in the surgical management should continue because mortality and morbidity are almost always surgically related. Finally, indications for a palliative procedure persist in a small number of patients with complex intracardiac anatomy not amenable to early repair.

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